Dietary Sodium and Potassium Intake Is Not Associated With Elevated Blood Pressure in US Adults With No Prior History of Hypertension

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The relationship between dietary sodium and potassium intake with elevated blood pressure (BP) levels is unclear. The authors examined the association between dietary sodium and potassium intake and BP levels in 6985 adults aged 18 years and older with no prior history of hypertension who participated in the National Health and Nutrition Examination Survey (2001–2006). After adjustment for age, sex, race, body mass index, diabetes, and estimated glomerular filtration rate, there was no association between higher quartiles of sodium or potassium intake with the risk of a BP >140/90 mm Hg or >130/80 mm Hg. There was also no relationship between dietary sodium and potassium intake with BP when systolic and diastolic BP were measured as continuous outcomes (P=.68 and P=.74, respectively). Furthermore, no association was found between combinations of sodium and potassium intake with elevated BP. In the US adult population without hypertension, increased dietary sodium or low potassium intake was not associated with elevated BP levels. *J Clin Hypertens (Greenwich).* 2014;16:418–423. ©2014 Wiley Periodicals, Inc.

Hypertension affects almost a quarter of the world's population and is predicted to increase by another 60% by the year 2025.[1] The complications of hypertension and associated comorbid conditions have a huge impact on health resources utilization and length and quality of life.[2] Hypertension contributes to atherosclerosis, and is independently associated with coronary artery disease (CAD) and cardiovascular disease morbidity and mortality.[3–5] Hypertension is a modifiable risk factor, as tighter control of blood pressure (BP) can prevent and delay the occurrence of complications. Several studies have shown a significant reduction in cardiovascular events with BP control.[6–8] It is believed, however, that <25% of hypertensive patients in the United States have their BP at goal.[9] Despite the advent of newer drugs and awareness, the prevalence of hypertension continues to be on the rise, in part due to the rising epidemics of obesity, diabetes, and kidney disease in the United States. Thus, there is a need for different strategies to decrease the incidence and prevalence of hypertension including lifestyle changes.

Dietary modification of sodium and potassium intake has long been adapted as a nonpharmacologic modality of treatment for elevated BP. Clinical guidelines recommend a low-sodium and high-potassium diet to reduce BP and potentially modify the risk and severity of complications.[10] The recommendation is supported by a recent meta-analysis of 13 randomized controlled trials of salt reduction in individuals with type 1 and 2 diabetes that found a large reduction in BP with salt restriction.[11] Multiple epidemiologic studies demonstrate lower BP levels in populations consuming high-potassium diets.[12,13] One of the largest meta-analyses of randomized controlled trials of potassium supplementation has shown that an increase in potassium intake of 0.78 gm (20 mmol) per day is associated with an average reduction of 4.9 mm Hg of systolic BP (SBP) and 2.7 mm Hg of diastolic BP (DBP) among hypertensive patients.[14] Studies also show an inverse association between intake of potassium and BP,[15] with such an effect being more pronounced in hypertensive patients.[16]

The relationship between dietary sodium intake and hypertension has been the subject of a continuing debate as it is difficult to show a clear relationship between sodium and BP in population-based studies.[17] Furthermore, the role of dietary sodium and potassium intake on the development of elevated BP in populations without a prior diagnosis of hypertension is unclear. We performed a cross-sectional study using the National Health and Nutrition Examination Survey (NHANES 2001–2006) to test the hypothesis that high dietary sodium intake and low dietary potassium intake is associated with an increased risk of elevated BP in US adults with no known history of hypertension.

METHODS

Study Population

NHANES is a population-based survey designed to assess the health and nutritional status of adults and children in the United States and is unique in that it combines interviews and physical examinations. The NHANES interview includes demographic, socioeconomic, dietary, and health-related questions, while the examination component consists of medical, dental, and physiological measurements, as well as laboratory tests.

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NHANES used a stratified, multistage sampling design, with oversampling of African Americans, Hispanics, and persons older than 60 years, in order to produce reliable statistics. We used data from 31,507 participants from NHANES 2001–2006. Data were weighted using the dietary weights as described in the statistical analysis for NHANES data. Participants were excluded if they had a history of hypertension, lacked data on dietary sodium and potassium intake, were missing data for the calculation of estimated glomerular filtration rate (eGFR) by the abbreviated Modification of Diet in Renal Disease formula (MDRD),[18] did not have measurements of SBP and DBP (n=21,279), or did not have positive weights for the analysis (n=3243). The final sample used in this study included 6985 adult participants.

Variable and Outcome

The independent variables of interest were dietary sodium and potassium intake. Dietary intakes were calculated from 24-hour dietary recalls that were retrieved from all NHANES examinees. All the interviews were conducted at the mobile examination centers by trained interviewers. Each examination room contained a standard set of measuring guides to help the participant estimate portion sizes. Data were collected on total nutrient intake and individual foods. Information on added salt (frequency and type) were obtained apart from detailed descriptions about food reported (ie, type, form, brand name, and amount consumed) and nutrients from each food. The primary outcome of interest was elevated BP levels. For the purpose of this analysis, we examined two clinically relevant BP categories: >140/90 mm Hg and >130/80 mm Hg. We also examined SBP and DBP as continuous outcomes. SBP and DBP were measured in a standard fashion and 3 readings were collected from each participant.

Baseline Demographic and Clinical Data

Demographic information of the participants was gathered through questionnaires. Race/ethnicity was broken into 4 categories: non-Hispanic white, non-Hispanic black, Mexican American, and other. Participants were defined as having diabetes when they reported taking medication for diabetes, had a fasting glucose concentration ≥126 g/dL, or reported being told by a physician they have diabetes. Body mass index (BMI) was calculated as weight in kilograms divided by the square of height in meters. eGFR was calculated using the 4-variable MDRD equation.[18]

Statistical Analysis

Dietary intake of sodium and potassium was studied in quartiles. Quartiles of sodium intake 1 through 4 were ≤2190, 2191 to 3142, 3143 to 4349, >4349 mg/d and quartiles of potassium intake 1 through 4 were ≤1771, 1772 to 2529, 2530 to 3450, >3450 mg/d. For the purpose of analysis, we used the lowest quartile as the reference group for sodium intake and the highest quartile as the reference group for potassium intake. Chi-square test was used to compare categorical data and analysis of variance was used to compare continuous variables across the quartiles of dietary sodium and potassium intake. Multivariate logistic regression models were used to examine the association between high-sodium and low-potassium intake and development of elevated BP levels. All multivariate analyses were adjusted for age, sex, race, diabetes, BMI, and eGFR. Median intakes of sodium and potassium were used to determine "high-" and "low-" intake groups. Four groups of sodium and potassium intake were defined as follows: high-sodium/high-potassium intake; high-sodium/low-potassium intake; low-sodium/high-potassium intake, and low-sodium/low-potassium intake. We then examined the relationship of combinations of sodium and potassium intake with development of high BP. Two-tailed values of $P<.05$ were considered statistically significant. However, due to the large sample size, the magnitudes of the odds ratios (ORs) were considered to be more important in determining the effect size. All statistical analyses were performed using SAS software, version 9.2 Proc Survey (SAS Institute, Cary, NC).

RESULTS

Baseline Characteristics

The mean (standard error [SE]) age and eGFR of the participants was 41.6±0.4 years and 91.1±0.6 mL/min/1.73 m^2, respectively. The mean (SE) dietary sodium and potassium intake were 3579.7±30.3 mg/d and 2813.7±28.8 mg/d, respectively. The mean (SE) SBP and DBP was 117.6±0.3 and 70.3±0.2 mm Hg, respectively. In this cohort, 646 (9.2%) patients had a BP >140/90 mm Hg and 1692 (24.2%) had a BP >130/80 mm Hg. Baseline characteristics of the participants across the quartiles of dietary sodium and potassium intake are shown in Table I. Participants in the highest quartile of sodium intake were more likely to be male, to be younger, to be non-Hispanic white, and to have higher BMI and eGFR than participants in the lower quartiles of sodium intake. Participants in the highest quartile of potassium intake were older, more likely to be male, more likely to be white, and had lower eGFR compared with patients in the lower quartiles of potassium intake.

Dietary Sodium and Potassium Intake and Elevated BP

Findings from the logistic regression analysis examining the relationship between sodium intake and elevated BP levels >140/90 mm Hg and >130/80 mm Hg are shown in Table II. in In the unadjusted analysis, when elevated BP was defined as >140/90 mm Hg, the second and fourth quartiles of sodium were protective for elevated BP compared with the lowest quartile of sodium intake with respective ORs of 0.73 (95% confidence interval [CI], 0.55–0.98; $P=.04$) and 0.72 (95% CI, 0.54–0.96;

TABLE I. Baseline Characteristics of Study Participants Across Quartiles of Dietary Sodium and Potassium Intake

	Sodium Intake, mg/d					Potassium Intake, mg/d				
Characteristics	≤2190	2191–3142	3143–4349	>4349	*P* Value	≤1771	1772–2529	2530–3450	>3450	*P* Value
Mean age (SD), y	43.6±0.5	42.4±0.6	41.9±0.5	38.9±0.6	<.01	39.3±0.5	42.1±0.6	42.3 ±0.6	42.1±0.6	.01
Male sex, %	31.0	39.1	49.8	73.5	<.01	30.5	41.4	52.7	70.0	<.01
Race, %										
Non-Hispanic black	11.5	10.9	6.8	9.0	<.01	15.1	10.4	7.3	5.8	<.01
Non-Hispanic white	67.8	71.8	75.1	75.7		65.3	70.1	77.1	77.0	
Mexican American	10.1	9.8	8.8	8.1		9.3	10.0	7.7	9.7	
Diabetes, %	3.3	3.6	3.3	2.5	.73	3.2	3.5	2.7	3.2	.78
BP >140/90 mm Hg, %	10.6	7.9	8.2	7.9	<.09	7.9	9.8	9.4	7.3	.21
BP >130/80 mm Hg, %	26.2	25.0	25.4	24.7	.91	22.8	27.1	26.2	24.7	.12
BMI, kg/m^2	26.7±0.2*	26.9±0.2*	27.5±0.2	27.5±0.2	.01	27.5±0.2	27.3±0.2	27.0±0.2	26.9±0.2	.18
eGFR, mL/min/1.73 m^2	90.0±0.9*	90.7±1.0	90.4±0.8	93.1±0.8	.01	93.2±0.7	91.0±0.9*	90.5±0.9*	89.9±0.8*	.01
SBP, mm Hg	117.7±0.6	117.3±0.4	117.9±0.5	117.4±0.4	.74	116.2±0.5	118.4±0.6*	118.5±0.5*	117.1±0.4	.01
DBP, mm Hg	70.1±0.4	69.8±0.4	70.8±0.4	70.4±0.4	.15	69.7±0.4	70.1±0.4	70.8±0.4	70.5±0.3	.08

Values are expressed as mean±standard error unless otherwise specified. Abbreviations: BMI, body mass index; BP, blood pressure; DBP, diastolic blood pressure; eGFR, estimated glomerular filtration rate; SBP, systolic blood pressure. *P <0.05 compared to the 1st quartile.

TABLE II. Odds Ratio (95% Confidence Interval) of Blood Pressure (BP) >140/90 mm Hg and >130/80 mm Hg by Quartiles of Dietary Sodium Intake.

	Sodium Intake, mg/d			
Elevated BP Level	≤2190	2191–3142	3143–4349	>4349
>140/90 mm Hg				
Unadjusted	1.00 (Referent)	0.73 (0.55–0.98)	0.75 (0.56–1.02)	0.72 (0.54–0.96)
Fully adjusted[a]	1.00 (Referent)	0.75 (0.54–1.04)	0.81 (0.56–1.16)	0.98 (0.71–1.37)
>130/80 mm Hg				
Unadjusted	1.00 (Referent)	0.94 (0.75–1.18)	0.96 (0.76–1.21)	0.93 (0.75–1.14)
Fully adjusted[a]	1.00 (Referent)	0.92 (0.71–1.19)	0.88 (0.67–1.15)	0.83 (0.63–1.09)

[a]Adjusted for age, sex, race/ethnicity, body mass index, estimated glomerular filtration rate, and history of diabetes.

P=.03). Similarly, in the unadjusted analysis, the third quartile of sodium intake was nearly significantly protective for elevated BP compared with the lowest quartile OR 0.75 (95% CI, 0.56–1.02; *P*=.06). After adjusting for variables that could independently affect BP levels including age, sex, race, BMI, history of diabetes and eGFR, there was no longer an association between sodium intake and BP level >140/90 mm Hg. Similarly, no association was found between sodium intake and BP >130/80 mm Hg in unadjusted or adjusted analyses (Table II). There was also no association between sodium intake and BP when SBP and DBP were examined continuously (adjusted β±SE −0.0001± 0.0001 [*P*=.68] and β±SE −0.00002±0.00006 [*P*=.74], respectively). In a sensitivity analysis, we examined the Institute of Medicine's cutoff for sodium intake in high-risk populations of <1500 mg/d vs >1500 mg/d. In unadjusted analysis, a sodium intake of >1500 mg/d was protective for elevated BP >140/90 mm Hg (OR, 0.79; 95% CI, 0.64–0.97; *P*=.03) but after adjusted for age, sex, race, BMI, history of diabetes, and eGFR, this relationship was no longer significant (OR, 0.93; 95% CI, 0.67–1.28; *P*=.65). A sodium intake <1500 mg/d was not protective for BP >130/80 mm Hg in unadjusted or adjusted analyses.

The relationship between dietary potassium intake and development of elevated BP levels is shown in Table III. There was no association between dietary potassium intake and elevated BP levels in unadjusted analysis. After adjustment for age, sex, race, BMI, history of diabetes, and eGFR, there was no association between potassium intake and elevated BP levels, defined as >140/90 mm Hg. When elevated BP level was defined as >130/80 mm Hg, patients in the second quartile of potassium intake had an increased risk of elevated BP compared with patients in the fourth quartile (adjusted OR, 1.35; 95% CI, 1.06–1.71; *P*=.01) (Table III). There was a trend towards an increased risk of elevated BP levels in patients in the first quartile of potassium intake but it did not reach statistical significance (adjusted OR, 1.24; 95% CI, 0.98–1.58; *P*=.08). There was also no association between potassium intake and BP when SBP and DBP were examined continuously (adjusted β±SE −0.0005±

TABLE III. Odds Ratio (95% Confidence Interval) of Blood Pressure (BP) >140/90 mm Hg and >130/80 mm Hg by Quartiles of Dietary Potassium Intake

	Potassium Intake, mg/d			
Elevated BP Level	≤1771	1772–2529	2530–3450	>3450
>140/90 mm Hg				
Unadjusted	1.08 (0.75–1.56)	1.37 (0.97–1.94)	1.31 (0.89–1.91)	1.00 (Referent)
Fully adjusted[a]	1.16 (0.78–1.70)	1.33 (0.92–1.91)	1.28 (0.84–1.94)	1.00 (Referent)
>130/80 mm Hg				
Unadjusted	0.90 (0.74–1.09)	1.13 (0.92–1.39)	1.08 (0.89–1.31)	1.00 (Referent)
Fully adjusted[a]	1.24 (0.98–1.58)	1.35 (1.06–1.71)	1.18 (0.93–1.49)	1.00 (Referent)

[a]Adjusted for age, sex, race/ethnicity, body mass index, estimated glomerular filtration rate, and history of diabetes.

TABLE IV. Odds Ratio (95% Confidence Interval) of Blood Pressure (BP) >140/90 mm Hg and >130/80 mm Hg by Combinations of Dietary Sodium and Potassium Intake

	Combinations of Dietary Intake			
Elevated BP Level	Low Sodium/Low Potassium	Low Sodium/High Potassium	High Sodium/Low Potassium	High Sodium/High Potassium
>140/90 mm Hg				
Unadjusted	1.00 (Referent)	0.94 (0.66–1.32)	0.80 (0.54–1.17)	0.86 (0.68–1.07)
Fully adjusted[a]	1.00 (Referent)	0.83 (0.56–1.21)	0.97 (0.63–1.49)	0.95 (0.74–1.21)
>130/80 mm Hg				
Unadjusted	1.00 (Referent)	1.16 (0.93–1.45)	1.07 (0.83–1.37)	1.00 (0.84–1.19)
Fully adjusted[a]	1.00 (Referent)	0.98 (0.76–1.26)	1.08 (0.83–1.42)	0.82 (0.66–1.01)

[a]Adjusted for age, sex, race/ethnicity, body mass index, estimated glomerular filtration rate, and history of diabetes.

0.0005 [*P*=.06] and β±SE −0.0001±0.00007 [*P*=.33], respectively).

We also examined the relationship of combinations of potassium and sodium intake with elevated BP levels >140/90 mm Hg and >130/80 mm Hg (Table IV). Median intake of daily dietary sodium (3142 mg/d) and potassium (2529 mg/d) were used to determine high- and low-sodium and potassium intake. After multivariate adjustment, none of the combinations of dietary sodium and potassium intake were associated with elevated BP levels >140/90 mm Hg or >130/80 mm Hg.

DISCUSSION

In this cross-sectional study of 6985 participants without a history of hypertension from NHANES 2001–2006, we did not find an association between dietary intake of sodium and potassium and the risk of elevated BP levels. Higher sodium intake either in isolation or in conjunction with low-potassium intake did not increase the odds of elevated BP.

Our results conflict with previous population-based studies showing an increased risk of elevated BP with high-sodium and low-potassium diets. For example, the SALTURK study examined both normotensive and hypertensive adults and found that each 800 mg increase of sodium intake per day was associated with an increase in SBP by 5.8 mm Hg.[19] Compared with our study, the mean sodium intake was much higher in the SALTURK study (7200 mg/d compared to 3580 mg/d), which could be the reason for conflicting results. However, other population-based studies have found no association between dietary sodium and potassium intake with BP. A study of 2391 men and women in the Netherlands found no association between sodium or potassium intake and BP estimated from a 1-week dietary recall.[20] A small study of healthy participants in Paraguay also did not find an association between elevated BP and dietary sodium or potassium intake.[21] A previous analysis using NHANES I data examined participants without a history of hypertension and found that higher intakes of sodium and potassium were associated with a lower not higher mean SBP.[22] Another study performed in NHANES I found no relationship between sodium and potassium intake and BP.[23] Our results confirm these findings in the later NHANES cohort 2001–2006. Compared with NHANES I, the 24-hour dietary recall information from the continuous NHANES cohort was collected by trained interviewers with the use of standard measuring guides to estimate intake. Hence, our estimates of intake are likely more accurate than those in NHANES I. Compared with our study, the mean (standard deviation) sodium and potassium intakes were much lower in NHANES I (1916±1158 mg/d and 1902±1012 mg/d, respectively). In the majority of these studies, including ours, there were few participants with stage II hypertension. Hence, it is possible that we did not have high

enough BPs to find an association between sodium and potassium intake and BP. Numerous interventional studies have shown that lowering sodium intake lowers BP in both hypertensive and normotensive adults.[11,24–26] Perhaps the reason large epidemiologic studies such as ours did not find an association between sodium and BP is that we examined sodium intake at the population level. A study by Ducher and colleagues examined normotensive adults followed for 2 years and found no association of sodium intake with BP at the population level, but, after conducting an analysis of statistical dependence between sodium intake and BP *within* individuals, they did find a correlation.

Previous studies have shown that a higher dietary sodium to potassium ratio plays an important role in the pathogenesis of hypertension independent of cardiovascular risk factors.[27] Similarly, the degree of BP reduction from potassium depends on the concurrent sodium intake, so the higher the sodium intake, the better the BP-lowering effect of increased potassium intake.[14] However, in our cohort, none of the combinations of sodium and potassium intake reduced the odds of elevated BPs. Our results further add to the controversy regarding the association of sodium and potassium intake with elevated BP.

A population-wide reduction in dietary sodium intake has been adapted as a prophylactic initiative to lower BP and cardiovascular events.[11,24–26] While studies have shown BP reduction with salt restriction,[11,24–26] the beneficial role of low-sodium intake has been questioned by other studies showing a higher risk of all-cause and cardiovascular mortality with low-sodium intake.[28] A study of 28,880 middle-age adults with hypertension found that both high and low levels of salt increased their risk of cardiovascular disease and death.[29] A large observational study of 2807 patients with type 1 diabetes, also found a nonlinear association of urinary sodium excretion with death, such that participants with the highest as well as the lowest urinary sodium excretion had an increased risk of death.[30] Low intake of sodium can lead to reflex activation of the renin-angiotensin-aldosterone system (RAAS) and sympathetic nervous system, as well as metabolic pathways resulting in increases in total cholesterol and low-density lipoprotein cholesterol.[31] Whether these effects of low-sodium intake explain the increased cardiovascular and overall mortality is not completely understood. Based on these observational studies, the Institute of Medicine reported that there was inconclusive evidence to support lowering sodium intake to <2300 mg/d in any population.[32] The committee identified methodological gaps in studies, especially population-based studies, as an issue that needs correction. Indeed, our results conflict with previous studies, likely as a result of differences in methodology. A standardized approach for measuring dietary sodium and potassium intake is needed. Ultimately, randomized controlled trials are needed in order to determine the effects of sodium intake on health outcomes, especially in high-risk populations such as those with cardiovascular and kidney disease.

STUDY LIMITATIONS

Our study has several limitations. First, no causal relationship between dietary intakes of sodium and potassium and BP levels can be established because of the observational design of the study. Second, we used dietary recall to estimate 24-hour dietary intake due to unavailability of data on urinary sodium and potassium excretion. Twenty-four–hour urinary sodium and potassium measurement is considered the gold standard method for estimation of dietary intake, and dietary recalls can underestimate sodium intakes. However, the high-quality survey methodology and meticulous attention given to sodium content of food in NHANES offsets some of the inherent problems associated with dietary recalls. Despite the likelihood that dietary recall can give an imprecise estimation of actual consumption, a uniform underestimation or overestimation of actual dietary intake by the 24-hour recall should not have affected our results.

STUDY STRENGTHS

Barring the above-mentioned shortcomings, our study also has several strengths. This is the first study, to our knowledge, examining the association between sodium and potassium intake and elevated BP in patients without a diagnosis of hypertension in NHANES 2001–2006. Secondly, NHANES uses uniform methods for dietary recall and BP measurement. The comprehensive NHANES dataset has information on various factors known to cause an elevation in BP such as chronic kidney disease, diabetes, age, sex, ethnicity, and BMI. Thus, we were also able to adjust for established risk factors known to cause an increase in BP. In addition, the NHANES cohort provides a good representation of the US civilian population, thus the results obtained from our study can be extrapolated to the noninstitutionalized US population.

CONCLUSIONS

This population-based cohort study fails to show an association between dietary intakes of sodium and potassium and levels of BP. These findings should be further examined through clinical trials specifically designed to examine the effects of dietary sodium and potassium intake on BP.

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